

Recent Tanning Bed Use

A Risk Factor for Melanoma

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Background: Individuals at increased risk of melanoma should use sun-protective measures to decrease their risk of developing melanoma.

Observation: We report a case of a 39-year-old patient with a *CDKN2A* mutation who developed 3 primary melanomas within a few years of initiating tanning bed use.

Conclusion: Intense UV exposure as an adult likely contributed to the development of additional primary melanomas in this individual.

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SUN OR UV RADIATION EXPOSURE is one of the principal causal factors in the development of cutaneous malignant melanoma. A growing body of evidence suggests that not only childhood but also adult exposure is related to risk of melanoma, even among those who tan well,^{1,2} and that use of tanning beds increases melanoma risk.^{3,4} Our case report highlights the importance of intense adult UV exposure in a genetically susceptible patient. We describe a patient with familial melanoma who developed numerous active nevi and 3 melanomas 2 ½ years after initiation of tanning bed use.

REPORT OF A CASE

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A 39-year-old white man with a medical history significant for 1 in situ and 11 invasive melanomas (**Figure 1**), dysplastic nevi, and a *CDKN2A* mutation was seen for his routine skin examination. His first melanoma was diagnosed when he was an adolescent; he was first evaluated by our group as a young adult. He had extensive recreational sun exposure in the past, having grown up in the south and frequently vacationed at the beach. He had not traveled to other sunny vacation areas. He was being observed regularly for over 650 nevi.

Unlike during other visits in the previous 6 years, the patient appeared deeply tanned and reported recent frequent tanning bed use at his health club. He denied erythema following exposure. By history, the patient stated that his nevi had not changed in number or appearance.

On physical examination, the patient's skin was difficult to evaluate because many nevi appeared to be a color similar to that of his tanned skin. Although several nevi had changed somewhat, no nevi were observed to have changed in a manner suggestive of melanoma, and biopsy was not indicated. Full-body overview photographs were taken of his skin and close-ups were taken of selected nevi. The patient was instructed on sun-protective measures and the importance of skin self-examinations, and he was urged to avoid tanning bed exposure.

Three months later, the patient returned for a scheduled follow-up visit. He reported continued regular tanning bed use several times a week. On examination, his skin appeared deeply tanned, and multiple nevi had increased in size and become more atypical. Four lesions were excised, and analysis of biopsy specimens revealed mild to moderate dysplasia. Pursuant to continued counseling, the patient discontinued his tanning bed use approximately 9 months after first use and

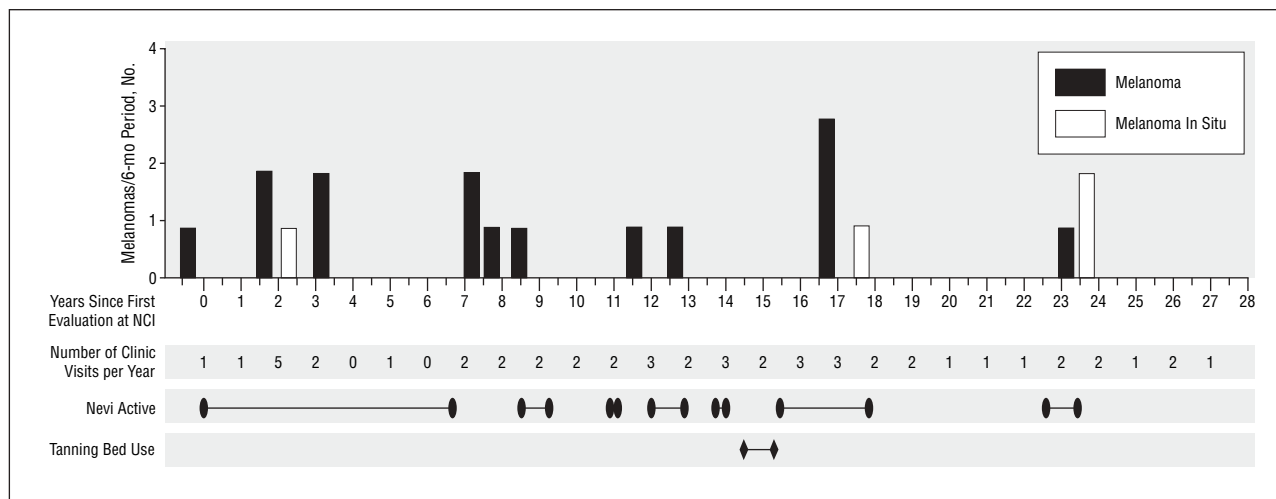


Figure 1. Timeline of the patient's course from our initial evaluation. The numbers of new invasive or in situ melanomas per 6-month interval are indicated by the vertical bars. Numbers of examinations per year are shown as numbers beneath each year interval. Periods of nevus activity are shown in horizontal bars by the time interval. The period of intense tanning bed exposure is also shown as a horizontal bar. NCI indicates National Cancer Institute.

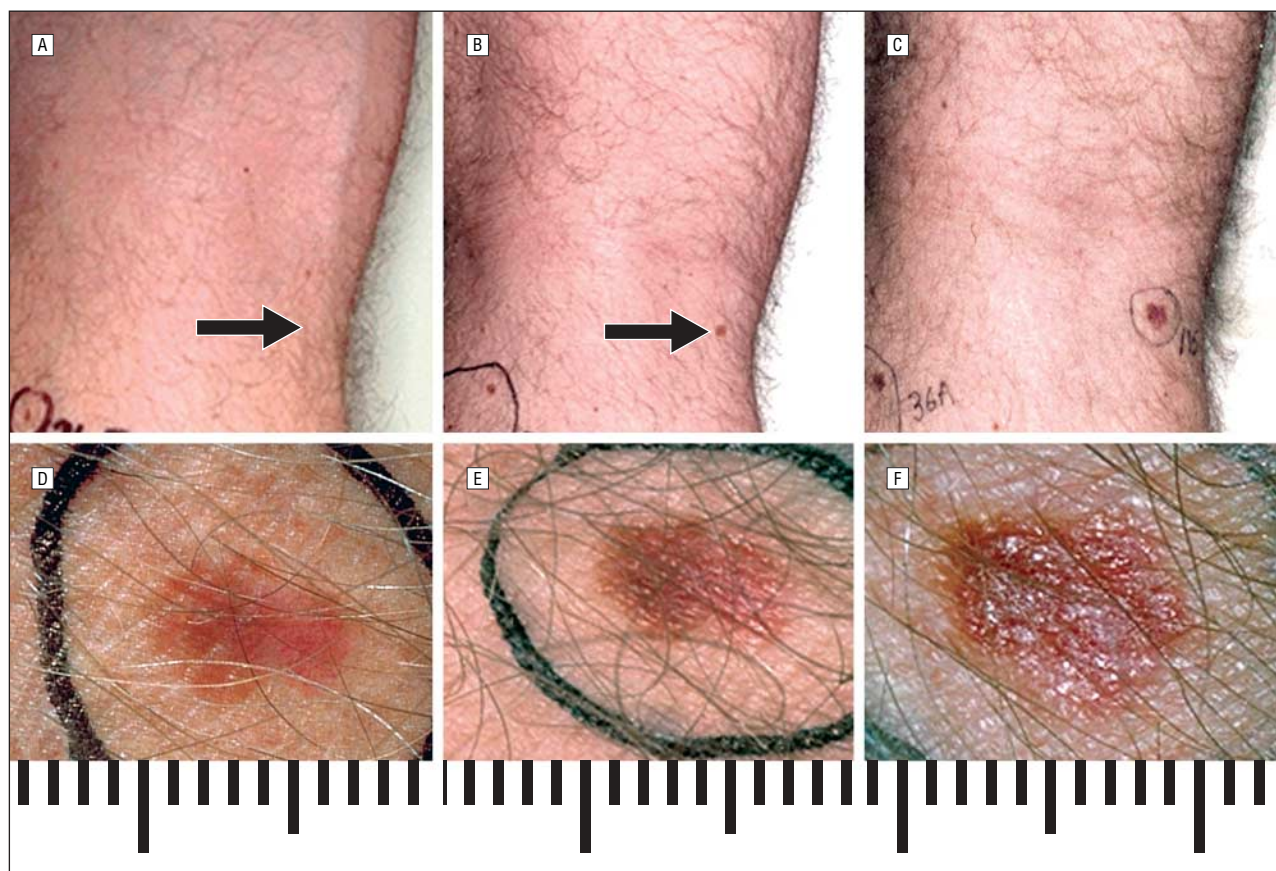


Figure 2. Lesion A evolution. A, Left popliteal fossa approximately 12 years prior to tanning bed use. A nevus is located superiorly, and a smaller, less visible nevus (arrow) is located directly below this lesion. B, Ten years later, the superior nevus is relatively unchanged, but the nevus located inferiorly (arrow) has enlarged and become more prominent. C, Approximately 2.5 years after the initiation of tanning bed use, the lesion has grown further. D, Close-up of the nevus taken 3.5 years after the photograph in panel B was taken. The lesion is no longer symmetric and has a new erythematous macule to the right. E, Enlargement of the erythematous portion has occurred over a 7-month period. F, Ten months later, the nevus has become substantially larger. This close-up, taken at the same time as panel C, and just prior to excision, reveals that the lesion borders are less distinct, and there is a fine scale overlying the entire lesion. A biopsy specimen revealed a 0.55-mm-thick malignant melanoma of the superficial spreading type with radial and vertical growth phase. Gradient lines in panels D through F represent millimeters.

several months after the most recent biopsy. Figure 1 provides a timeline of the patient's tanning bed use and the activity of his nevi over time. Approximately 2 years after he had initiated tanning bed use, 2 nevi that had changed

in a manner suggestive of melanoma were excised, and analysis revealed severe dysplasia.

Six months later, he developed numerous new dark nevi (<4 mm), particularly on the abdomen. In addition, there



Figure 3. Evolution of a nevus on the patient's shoulder. A, Routine overview of the shoulder taken approximately 1 month prior to the initiation of tanning bed use. Note the nevus located infraclavicularly (arrow). B, The same region approximately 4 months later when the patient was quite tan. C, Two and a half years later, the nevus on the shoulder had become more prominent and irregular. D, Close-up of this asymmetric lesion, which measures approximately 7 mm in diameter, demonstrates the erythematous base, areas of hypopigmentation, and indistinct borders. The lesion was excised, and analysis revealed a 0.45-mm-thick superficial spreading melanoma, radial growth phase only.

were 3 specific nevi of concern for melanoma (A, B, and C) located in the popliteal fossae and clavicular region, and these were excised several weeks after discovery. **Figure 2** illustrates lesion A in the left popliteal fossa. Lesion A was under observation as a clinically dysplastic nevus and was excised because of increasing asymmetry and diameter over 3 examinations. Histologic examination revealed a superficial spreading malignant melanoma with radial and vertical growth phase, Breslow depth, 0.55 mm. Lesion B in the right popliteal fossa had not previously been observed. It was found to be a *de novo* superficial spreading malignant melanoma, radial growth phase only, Breslow depth, 0.37 mm.⁵ Lesion C (**Figure 3**) was located in the clavicular region. A small nevus had been observed prior to tanning but was not considered to be clinically dysplastic. The lesion was subsequently excised as suggestive of melanoma owing to increasing size and asymmetry as well as areas of hypopigmentation and erythema. This lesion was a superficial spreading malignant melanoma, radial growth phase only, Breslow depth, 0.45 mm.

After the 3 melanoma excisions, the patient routinely returned for follow-up. Three months after the excision of the lesions, a moderately dysplastic nevus was excised; 1 year after that, a melanoma *in situ* was removed. He continued to adhere to sun-protective measures and denied further tanning bed use. Most of his nevi either remained stable or began to fade over the next several months. In subsequent visits, continued regression of the nevi was noted, and many lesions disappeared. Nine years after the intense UV exposure in the tanning bed, 1 invasive and 2 *in situ* melanomas were excised. To date,

it has been 12 years since the patient used a tanning bed and 4 years since his last melanoma. He continues to use sun-protective behaviors and has not tanned. Most of his atypical nevi have faded and regressed. He developed several new small symmetric nevi, most of which have also faded or disappeared.

COMMENT

The rising incidence of melanoma in the United States is at least partly a result of increased sun exposure.⁶ The likelihood of developing melanoma depends on host characteristics, environmental exposures, and genetic predisposition. The individual described herein has multiple risk factors for melanoma, including mutation in a major melanoma susceptibility gene, *CDKN2A*, multiple dysplastic nevi, previous multiple primary melanomas, and past sun exposure. The development of new melanomas shortly after intense tanning bed use is of interest; we have documented development of a *de novo* melanoma and 2 melanomas arising in previously unremarkable nevi over a short time interval. In addition, 6 other nevi were excised because of worrisome changes within the same time span.

This individual had substantial sun exposure prior to the tanning bed exposure. He is thus very similar to many individuals using tanning parlors. Those who exhibit sun-seeking behavior will always have complex exposures with mixes of different UV action spectra.^{3,4,7} It is virtually impossible to precisely quantify total life exposure, but rou-

tine tanning bed use over many months represents an intense burst of UV exposure. Rapid change in multiple nevi and the development of new lesions shortly after this are likely related to activation of the melanocytic system. Although early sun exposure is important generally in the development of melanoma,¹ and was specifically in this individual (since he developed his first melanoma at age 15 years), the short latency of these reported lesions suggests that adult exposure also contributes to the development of melanoma. This observation is consistent with a growing body of evidence from multiple epidemiologic studies.^{2,8,9}

Obviously, this individual also has genetic predisposition to developing melanoma, which may have affected the length of the latency. Data from transgenic mouse models suggest that in genetically susceptible mice irradiated as neonates, deficiency of *ink4a/arf* decreases the latency of melanomas.^{10,11} It is clear from studies of high-risk families with mutations in *CDKN2A*, however, that additional factors affect risk of melanoma.¹²⁻¹⁴

Our findings represent observations from a single patient, and it would be inappropriate to generalize to all genetically susceptible patients or to the general population. However, this case illustrates that intense adult UV exposure may be important in high-risk individuals. It is unusual for such people under close surveillance to have artificial UV exposures as described herein. We believe that this report could be a useful illustration for health care providers in educating high-risk individuals.

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REFERENCES

- Whiteman DC, Whiteman CA, Green AC. Childhood sun exposure as a risk factor for melanoma: a systematic review of epidemiologic studies. *Cancer Causes Control*. 2001;12:69-82.
- Fears TR, Bird CC, Guerry D, et al. Average midrange ultraviolet radiation flux and time outdoors predict melanoma risk. *Cancer Res*. 2002;62:3992-3996.
- Gallagher RP, Spinelli JJ, Lee TK. Tanning beds, sunlamps, and risk of cutaneous malignant melanoma. *Cancer Epidemiol Biomarkers Prev*. 2005;14:562-566.
- Autier P. Perspectives in melanoma prevention: the case of sunbeds. *Eur J Cancer*. 2004;40:2367-2376.
- Tucker MA, Fraser MC, Goldstein AM, et al. A natural history of melanomas and dysplastic nevi: an atlas of lesions in melanoma-prone families. *Cancer*. 2002;94:3192-3209.
- Jemal A, Devesa SS, Hartge P, Tucker MA. Recent trends in cutaneous melanoma incidence among whites in the United States. *J Natl Cancer Inst*. 2001;93:678-683.
- Lazovich D, Forster J. Indoor tanning by adolescents: prevalence, practices and policies. *Eur J Cancer*. 2005;41:20-27.
- Pfahlerberg A, Kolmel KF, Gefeller O. Timing of excessive ultraviolet radiation and melanoma: epidemiology does not support the existence of a critical period of high susceptibility to solar ultraviolet radiation-induced melanoma. *Br J Dermatol*. 2001;144:471-475.
- Robsahm TE, Tretli S. Cutaneous malignant melanoma in Norway: variation by region of residence before and after the age 17. *Cancer Causes Control*. 2001;12:569-576.
- Noonan FP, Recio JA, Takayama H, et al. Neonatal sunburn and melanoma in mice. *Nature*. 2001;413:271-272.
- Recio JA, Noonan FP, Takayama H, et al. *Ink4a/Arf* deficiency promotes ultraviolet radiation-induced melanomagenesis. *Cancer Res*. 2002;62:6724-6730.
- Bishop DT, Demenais FM, Goldstein AM, et al. Geographic variation in the penetrance of *CDKN2A* mutations for melanoma. *J Natl Cancer Inst*. 2002;94:894-903.
- Goldstein AM, Martinez M, Tucker MA, Demenais F. Gene-covariate interaction between dysplastic nevi and the *CDKN2A* gene in American melanoma-prone families. *Cancer Epidemiol Biomarkers Prev*. 2000;9:889-894.
- Chaudru V, Chompret A, Bressac-de Paillerets B, Spatz A, Avril M-F, Demenais F. Influence of genes, nevi, and sun sensitivity on melanoma risk in a family sample unselected by family history and in melanoma-prone families. *J Natl Cancer Inst*. 2004;96:785-795.